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In Vitro Pulmonary Toxicity of Metal Oxide Nanoparticles

Cupi, Denisa; Dreher, Kevin

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CONTACT (NAME ONLY): Kevin Dreher

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Abstract

TITLE: *IN VITRO* PULMONARY TOXICITY OF METAL OXIDE NANOPARTICLES

AUTHORS (LAST NAME, FIRST NAME): Cupi, Denisa[1]; Dreher, Kevin[2]

SPONSOR NAME: None

INSTITUTIONS (ALL): 1. Technical University of Denmark, Kongens Lyngby, Denmark.
2. US EPA, Research Triangle Park, NC, United States.

ABSTRACT BODY: Nanomaterials (NMs) encompass a diversity of materials with unique physicochemical characteristics which raise concerns about their potential risk to human health. Rapid predictive testing methods are needed to characterize NMs health effects as well as to screen and prioritize NMs for comprehensive toxicological assessments. BEAS2B human bronchial epithelial cells were employed to assess the *in vitro* pulmonary toxicity of 4 TiO2 and 4 CeO2 particles varying is size (6 - 1288nm) and crystalline structure. Exposures were conducted over several concentrations for each endpoint examined. No BEAS2B cytotoxicity was observed for any particle following a 24hr exposure to concentrations up to100 µg/ml. The ability of TiO2 and CeO2 particles to induce inflammation and oxidative stress was assessed by gene induction using RT-PCR. At 50 µg/ml maximal IL-8 and IL-6 gene induction by TiO2 and CeO2 NMs (6 - 8nm) was observed at 6hr and 24hr post-exposure, respectively. Smaller TiO2 and CeO2 NMs induced greater induction of IL-8 and IL-6 mRNA levels compared to larger sized particles. CeO2 8nm NMs produced the greatest induction of IL-8 and IL-6 mRNA levels. At 50 µg/ml all TiO2 and CeO2 particles induced similar increases in HO-1 mRNA levels at 6hr and 24hr post-exposure, respectively. The pattern of HO-1 gene induction was inconsistent with a role of oxidative stress in metal oxide induced BEAS2B cytokine gene expression. Pretreatment of BEAS2B cells with IKK inhibitor III BMS-345541 completely inhibit 25nm TiO2 and 69nm CeO2 NM induction of IL-8, IL-6 and HO-1 gene expression indicating a role of NFκB in these responses. A cell-based ELISA for NFκB p65 phosphorylation revealed rapid Ser536 phosphorylation in BEAS2B cells following exposure to 50 µg/ml of TiO2 and CeO2 NMs with sizes $\geq$25nm. Results demonstrate the ability to employ *in vitro* methods to assess NM induced pulmonary toxicity. Many of the responses were found not to be totally dependent on NM size/surface area suggesting composition and surface properties play a role in mediating NM toxicity. This abstract does not reflect EPA policy.